tients. Histologically, the average amount of parathyroid parenchyma in FHH is 300 percent of that in normal parathyroid glands but considerably less than that in typical primary hyperparathyroidism.[5] Incidence of persistence or recurrence of hypercalcemia is higher in patients with FHH after parathyroidectomy than in typical primary hyperparathyroidism.

As the condition has been recognized only during the last four to five years, the best treatment for these patients remains unclear.[6] The finding of hypocalciuric hypercalcemia in several relatives favors the diagnosis of FHH. In these patients no further surgical treatment for hypercalcemia per se is indicated. The patient described in this report continues to be asymptomatic and her calcium levels have ranged from 11.5 to 12 mg per dl. No surgical intervention was recommended to her. All patients suspected of having primary hyperparathyroidism should routinely have 24-hour urinary calcium test and renal calcium to creatinine clearance ratio determination to rule out FHH. This should be done particularly if the hyperparathyroidism is diagnosed in an asymptomatic patient. If values are consistent with those seen in FHH, then family screening is in order.

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Acute Polyarthritis and Infectious Mononucleosis

JEFFREY D. URMAN, MD
Redwood City, California

ARTHUR M. BOBROVE, MD
Palo Alto, California

INFECTIOUS MONONUCLEOSIS (IM) is a common illness with protean manifestations ranging from subclinical infection to severe disease affecting many different organ systems. Arthralgias may occur in approximately 5 percent to 10 percent of patients.[1] Well-documented synovitis has rarely been reported. Adebonojo[2] described a 7-year-old girl with infectious mononucleosis in whom an acute inflammatory monarthritis of the ankle joint developed several days after the onset of a sore throat. This report describes an adolescent boy with severe symptoms of IM in whom an acute inflammatory polyarthritis developed.

From the Department of Internal Medicine, Redwood Medical Clinic, Redwood City, California, and the Division of Rheumatology, Department of Internal Medicine, Palo Alto Medical Clinic, Palo Alto, California.

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Reprint requests to: Jeffrey D. Urman, MD, Redwood Medical Clinic, 2900 Whipple Avenue, Redwood City, CA 94062.

Report of a Case

A 16-year-old white boy developed an intensely pruritic, erythematous macular rash over his entire body. Two days later he had complaints of anorexia, fatigue, fever, severe sore throat, swelling of his hands and eyelids and photophobia. He had taken no medications before the onset of the illness. On physical examination his temperature was 38.6°C (101.5°F). There was pronounced injection of the conjunctivae, a fiery red inflammation of the oral mucosa and enlarged inflamed tonsils without exudate. There was mild cervical lymphadenopathy. The liver and spleen were not felt to be enlarged. There was a diffuse macular rash.

Initial laboratory studies showed a leukocyte count of 17,700 per cu mm with a leftward shift. There were only 4 percent lymphocytes, none of which were thought to be atypical. Wintrobe sedimentation rate was 24 mm per hour. Throat culture was negative for beta-hemolytic streptococcus and mononucleosis spot test was positive. Liver function studies were abnormal with moderate elevations in the serum aspartate aminotransferase (formerly serum glutamic oxaloacetic transaminase, SGOT) and serum alanine aminotransferase (formerly serum glutamic pyruvic transaminase, SGPT) and pronounced elevation in the alkaline phosphatase to 429 IU (30 to 100 IU). The lactic dehydrogenase (LDH) and bilirubin determinations were normal and there was no evidence of hepatitis B antigenemia. Complement-fixing antibody titers to herpes simplex,

adenovirus, mumps, cytomegalovirus, influenza and respiratory syncytial virus were each less than 1:8. Epstein-Barr virus (EBV) capsid antibody titer by the indirect fluorescent antibody technique was 1:256.

The patient was admitted to Sequoia Hospital (Redwood City) and begun on a regimen of prednisone, 60 mg daily taken by mouth. He improved rapidly and was discharged from hospital on the fourth day; prednisone administration was stopped three days later. Liver function studies showed improvement but there was not yet a return to normal.

One week after discharge the patient reentered the hospital with complaints of anorexia, fatigue, photophobia and pain in the elbows, wrists and knees. His temperature was 40°C (104°F). On examination he had enlarged lymph nodes and hepatosplenomegaly. Bilateral nummular keratitis had developed.[3] There was tenderness in both temporomandibular joints and in the left shoulder, and swelling and tenderness involving the left wrist, right elbow and fourth proximal interphalangeal joint of the right hand. There was a large effusion in the right knee. Thirty milliliters of cloudy yellow synovial fluid with poor viscosity was aspirated from the right knee. The fluid showed a leukocyte count of 21,000 per cu mm, with 86 percent polymorphonuclear leukocytes and 14 percent mononuclear cells. Gram stain and culture of the fluid were negative for pathogens. Other laboratory results showed a peripheral blood leukocyte count of 14,200 per cu mm with 93 percent polymorphonuclear leukocytes and 7 percent lymphocytes, none of which were atypical. There was persistent mild elevation in liver function studies. Analysis of urine showed no abnormalities. RA latex was negative. Fluorescent antinuclear antibody was present at low titer, 1:20, diffuse, and the C3 complement was moderately elevated. Blood cultures were consistently negative.

Repeat antibody titers for the above-mentioned viruses remained consistently less than 1:8. The EBV antibody titer, however, had increased to 1:2,048. Cryoglobulins were detected in his serum. HLA-B27 antigen was absent. Electrocardiogram showed diffuse ST-segment elevation consistent with acute pericarditis and a liver-spleen scan showed massive hepatosplenomegaly.

Treatment with prednisone was resumed at 60 mg a day. His symptoms lessened and results of laboratory tests returned toward normal ranges. The dose of prednisone was tapered off over a two-month period. He has remained free of rheumatic symptoms after three years of follow-up.

Comments

Rheumatic complaints may accompany several viral infections, most notably rubella, hepatitis B infections, mumps and varicella.[4] Arthralgias and arthritis accompanying infectious mononucleosis have been reported rarely but probably occur more frequently than appreciated.[4] Arthritis accompanying rubella and hepatitis B is thought to be on an immunologic basis resulting from deposition of antigen-antibody complexes in the synovial membrane. Infectious mononucleosis is often associated with increased immune reactivity causing a transient occurrence of various autoantibodies and cryoprecipitates.[5,6] Circulating immune complexes containing EBV antigen and complement components have been detected in patients with urticarial rash complicating infectious mononucleosis.[7] The detection of cryoprecipitates in our patient suggests their possible implication in his acute inflammatory polyarthritis. Unfortunately, analysis of synovial fluid did not include determinations of immune complexes or complement levels.

Our patient had severe disease with high fever and evidence of pericarditis and hepatitis in addition to the polyarthritis. He lacked two of the most characteristic laboratory findings in infectious mononucleosis (that is, lymphocytosis and increased numbers of atypical lymphocytes). Several blood counts actually showed lymphocytopenia. Lymphocytopenic infectious mononucleosis tends to be a more severe clinical illness.[1,8] The diagnosis in our patient was confirmed by both a positive mononucleosis spot test and a substantial rise in the convalescent antibody titer to EBV, a more specific test. Antibody titers to six other viruses showed no rise.

The 7-year-old girl with infectious mononucleosis in the report by Adebonojo had painful swelling of the left ankle, which on aspiration yielded inflammatory synovial fluid and responded within a week to aspirin and bed rest. In a recent review of viral arthritis, three patients with infectious mononucleosis and joint involvement who had not been previously reported were briefly discussed.[9] One had symmetrical involvement of the knees, wrists and small joints of the hands. The second had synovitis of the knees and the third had monarthritis involving a knee. The

synovial fluid was mildly inflammatory and yielded mostly mononuclear cells in two of the three patients.

Recently Pollack and co-workers described a 57-year-old man with heterophil-negative IM who presented with monarthritis of the knee.[10] Arthrocentesis yielded inflammatory synovial fluid with a preponderance of polymorphonuclear leukocytes. The patient was described as being severely ill and lacked atypical lymphocytes, which is similar to our patient.

Most patients with viral arthritis respond to nonsteroidal anti-inflammatory agents. Prednisone had been given to our patient because of the severity of his disease and the extensive organ system involvement. The arthritis responded promptly and completely in seven days.

Recently EBV has been implicated as possibly playing a role in causing rheumatoid arthritis. This stems from observations that patients with rheumatoid arthritis tend to have higher than normal antibody titers to two distinct EBV-associated antigens (rheumatoid arthritis nuclear antigen and Epstein-Barr nuclear antigen) present on EBV-infected lymphoblastoid cells.[11] Thus, EBV may prove to be associated with chronic arthritis.

Summary

Arthralgias occasionally accompany infectious mononucleosis (IM), but the occurrence of unmistakable arthritis is considered rare. We describe a teenaged boy with IM in whom acute inflammatory polyarthritis developed in the course of his disease. The patient had positive heterophilic antibodies but lacked atypical lymphocytes in his blood.

The EBV can thus be responsible for an inflammatory arthritis and should be added to the growing list of viruses known to be associated with this complication. This can be of particular importance in atypical cases of arthritis when the diagnosis of IM is not readily apparent.

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Postoperative Infection of an Aortic Prosthesis With *Achromobacter xylosoxidans*

DAVID A. OLSON, MD
PAUL D. HOEPRICH, MD
Sacramento, California

Achromobacter xylosoxidans is a Gram-negative bacillus that is motile (with peritrichous flagellae), nonfermentive and capable of catabolizing xylose but not lactose, maltose, mannitol or sucrose. It was originally isolated from human ear discharge specimens in 1971[1] and has been found in specimens from several other sites, including ventricular cerebrospinal fluid (CSF), pleural fluid, peritoneal fluid, urine, feces and swabs of the eye, ear and pharynx.[2] The natural habitat of *A xylosoxidans* is not known, but it may be a water bacterium, having been isolated from a swimming pool.[3] It has also been implicated in nosocomial infections by isolation from chlorhexidine solutions.[4] We report the case of a patient with infection of an aortic vascular prosthesis caused by *A xylosoxidans* to record the extent of pathogenicity of this microorganism. At the same time, this case shows the difficulty of achieving bactericidal therapy and chronicles the suppression

From the Section of Infectious and Immunologic Diseases, Department of Internal Medicine, University of California, Davis, School of Medicine.

Submitted February 24, 1981.

Reprint requests to: Paul D. Hoeprich, MD, Section of Infectious and Immunologic Diseases, UCD Professional Building, 4301 X Street, Sacramento, CA 95817.